Hypohydration does not impair skeletal muscle glycogen resynthesis after exercise

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NEUFER, P. DARRELL, MICHAEL N. SAWKA, ANDREW J. YOUNG, MARK D. QUIGLEY, WILLIAM A. LATZKA, AND LESLIE LEVINE. *Hypohydration does not impair skeletal muscle glycogen resynthesis after exercise.* J. Appl. Physiol. 70(4): 1490–1494, 1991.—The purpose of this investigation was to examine the effects of moderate hypohydration (HY) on skeletal muscle glycogen resynthesis after exhaustive exercise. On two occasions, eight males completed 2 h of intermittent cycle ergometer exercise (4 bouts of 17 min at 60% and 3 min at 80% of maximal O_2 consumption/10 min rest) to reduce muscle glycogen concentrations (control values 711 ± 41 μmol/g dry wt). During one trial, cycle exercise was followed by several hours of light upper body exercise in the heat without fluid replacement to induce HY (−5% body wt); in the second trial, sufficient water was ingested during the upper body exercise and heat exposure to maintain euhydration (EU). In both trials, 400 g of carbohydrate were ingested at the completion of exercise and followed by 15 h of rest while the desired hydration level was maintained. Muscle biopsy samples were obtained from the vastus lateralis immediately after intermittent cycle exercise (T1) and after 15 h of rest (T2). During the HY trial, the muscle water content was lower ($P < 0.05$) at T1 and T2 (288 ± 9 and 265 ± 5 ml/100 g dry wt, respectively; NS) than during EU (313 ± 8 and 301 ± 4 ml/100 g dry wt, respectively; NS). Muscle glycogen concentration was not significantly different during EU and HY at T1 (200 ± 35 vs. 251 ± 50 μmol/g dry wt) or T2 (452 ± 34 vs. 491 ± 35 μmol/g dry wt). These data indicate that, despite reduced water content during the first 15 h after heavy exercise, skeletal muscle glycogen resynthesis is not impaired.

carbohydrate metabolism; dehydration; muscle water; recovery from exercise

AFTER MUSCULAR ACTIVITY that depletes glycogen stores, glycogen resynthesis occurs in both skeletal muscle and liver, and glycogen concentration is usually observed to have returned to preexercise levels within 24 h after the activity (6, 15, 16). Incomplete replenishment of skeletal muscle glycogen during recovery from strenuous exercise has been attributed to insufficient carbohydrate intake and/or inadequate time for resynthesis (4, 6, 15). Little attention has been given to the possible influence of body water availability on muscle glycogen resynthesis. The binding of water with glycogen has been demonstrated in both liver and muscle, with estimates ranging from ~540 to 720 μl water · mmol glucose^{-1} · kg wet muscle^{-1} (20, 22). Thus, with a decrease in muscle glycogen concentration of 80 mmol glucose/kg wet muscle in ~10 kg of previously active muscle, it may be estimated that ~500 ml of water are released and thus could be required for the restoration of preexercise glycogen levels. Moreover, elite endurance athletes, whose capacity for glycogen storage may be >150 mmol glucose/kg wet muscle, may store as much as 1 liter of water in skeletal muscle. It is not known whether the amount of water available is a limiting factor for complete muscle glycogen resynthesis after exhaustive exercise.

It is not unusual for industrial workers, military personnel, and athletes to incur a 3–8% reduction in body weight (from water loss) during exercise in the heat (7, 10, 26). This body water deficit reduces an individual's ability to perform aerobic exercise primarily by acting on the cardiovascular and thermoregulatory systems (18, 25, 27, 28). The availability of water for muscle glycogen resynthesis might be compromised when prior exercise in the heat results in hypohydration. When hypohydration is mediated by sweating, the plasma becomes hyperosmotic, and this solute excess creates an osmotic gradient to move fluid from the intracellular to the extracellular space to defend plasma volume (27). As a result, sweat-mediated hypohydration results in water loss from both the intracellular and extracellular fluid spaces. Nose and colleagues (21) have shown that thermal-induced hypohydration results in water redistribution from extra- and intracellular spaces of primarily skeletal muscle and skin but not vital organs (e.g., liver and brain). Therefore it is reasonable to postulate that moderate hypohydration (sweat mediated) might reduce water content in the skeletal muscle in intracellular spaces and limit glycogen resynthesis.

The purpose of this investigation was to examine the effects of moderate hypohydration on skeletal muscle glycogen resynthesis after exhaustive exercise. It was hypothesized that hypohydration would reduce muscle water content and impair muscle glycogen resynthesis. Implicit in this hypothesis is the notion that water has a permissive role in muscle glycogen synthesis. From a practical standpoint, an inability to resynthesize muscle glycogen might provide an additional mechanism by which hypohydration decreases an individual's ability to perform prolonged exercise (1, 11, 28).

METHODS

Subjects. Eight men volunteered and provided written consent to participate in this study after being informed of the associated experimental requirements and possible risks. These men were all involved in a daily exercise program before the study, although none were highly

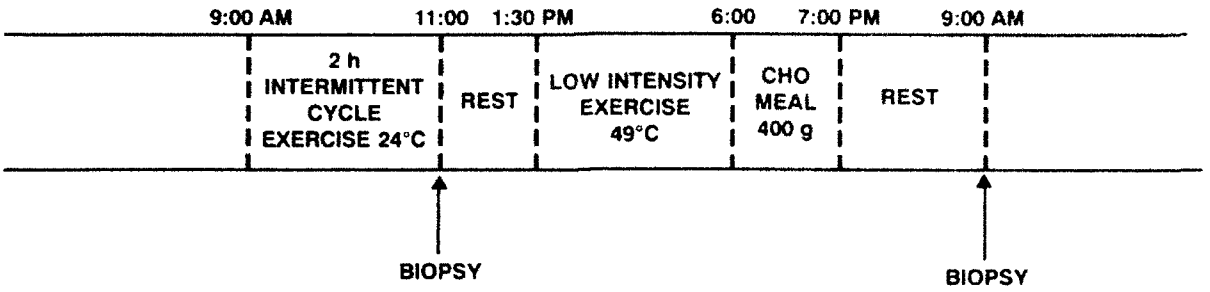

FIG. 1. Schematic of experimental design. CHO, carbohydrate.

trained athletes. Two weeks before experimental testing, each subject's percent body fat was determined by hydrostatic weighing, and maximal oxygen uptake ($\dot{V}O_{2\,max}$) was determined by a modification of the protocol described by Kamon and Pandolf (14). Nude body weights were obtained every morning before breakfast during the 2 wk before experimental testing. These measurements were used to establish baseline body weight, representing euhydration for each subject. The characteristics of the subjects were age 19 ± 1 (SE) yr, body fat 16.5 ± 1.6%, and $\dot{V}O_{2\,max}$ 3.77 ± 0.17 l/min (51 ± 3 ml · kg^{-1} · min^{-1}).

Experimental design. The subjects completed two trials separated by at least 1 wk. Each trial required ~26 h to complete. In one trial, after an exercise-dehydration protocol, glycogen resynthesis was determined during an overnight rest in which the subjects maintained a 5% hypohydrated state (HY trial). In a second trial, the subjects followed a similar protocol while maintaining euhydration (EU trial). Figure 1 provides a summary of the experimental design. During both trials, the subjects reported to the laboratory at 9:00 A.M. after an overnight fast and completed 2 h of intermittent cycle ergometer exercise in a temperate environment (23°C). In an effort to maximize glycogen depletion within the quadriceps muscle group, the 2 h of exercise were composed of four 30-min segments that each consisted of 17 min at 60% $\dot{V}O_{2\,max}$, 3 min at 80% $\dot{V}O_{2\,max}$, and 10 min of rest. A postexercise muscle sample was then obtained from the vastus lateralis muscle. The time at which this sample was obtained was designated as T1. During the HY trial, the subjects were not permitted to consume any fluids, whereas in the EU trial, fluid was ingested ad libitum during the cycle exercise. After exercise, the subjects were allowed to rest for ~1.5 h.

In the afternoon of each trial, the subjects returned to the laboratory to complete several hours of heat exposure and low-intensity exercise in a hot environment (49°C, 20% relative humidity). The exercise consisted of self-paced low-intensity arm-crank ergometer and rowing ergometer exercise with legs locked in the extended position. These exercise modes were selected in an effort to elicit sweating but minimize further activity of the quadriceps muscle groups. During the HY trial, the subjects continued with passive heat exposure and low-intensity exercise in the heat until they achieved a 5% reduction in body weight. The majority of weight loss was achieved by resting in a sauna. During the EU trial, the subjects were exposed to a similar amount of heat and exercise but were given sufficient volume of fluid to replace the body water loss incurred during the exercise-heat exposure. This phase of the protocol was completed by 5:00 P.M. for both trials.

At 6:00 P.M., the subjects consumed a 400-g carbohydrate meal consisting of pastries, candy, and concentrated glucose polymer drink. The subjects rested during the night in a comfortable environment. During the EU trial, fluid was given to each subject in sufficient volume to maintain baseline body weight, whereas during the HY trial subjects were instructed not to consume any fluids overnight. At ~9:00 A.M. the following morning (designated as T2), a final muscle sample was taken from the vastus lateralis. To further document the subjects' hydration level during the experiments, venous blood samples were obtained while they rested before the T2 biopsy. In addition, resting blood samples were obtained on a separate control day. Resting muscle glycogen levels were also assessed on a separate control day.

Physiological and biochemical measurements. The subjects' $\dot{V}O_{2\,max}$ was determined during cycle ergometer exercise with respiratory measurements made by use of an automated system (Sensormedic Horizon MMC). Subjects exercised for 5 min at a power output predicted to elicit each individual's $\dot{V}O_{2\,max}$ based on a previous submaximal bout. Subsequent 5-min exercise bouts were performed on successive days at power output levels set 15 W higher than the power output completed the previous day. O_2 uptake values were accepted as maximal when the difference was <75 ml O_2/min between power output differences of 15 W.

Muscle samples were obtained by biopsy of the vastus lateralis (1). Muscle samples were obtained from opposite thighs at T1 and T2; during the second trial, biopsy sites were at least 5 cm from sites used in the first experiment. After the biopsy, muscle samples were immediately dissected free of connective tissue, cut into 10- to 15-mg pieces, and repeatedly weighed (±0.01 mg) at timed intervals to allow correction for water evaporation during tissue handling (8). Muscle samples were frozen and stored in liquid N_2 for subsequent analysis of glycogen and water content. After being freeze-dried (72 h),

TABLE 1. *Body weight and blood constituents in control conditions and at completion of EU and HY trials*

	Body Wt, kg	Blood Parameters		
		Hemoglobin, g/dl	Plasma protein, g/dl	Osmolality, mosmol/kg
Control	73.2±3.8	14.9±0.4	8.1±0.2	281±2
EU	72.2±3.8	16.1±0.5*	8.2±0.2	277±1
HY	69.7±3.9*	17.0±0.3*	9.1±0.2*	294±1*

Values are means ± SE; n = 8 men. EU, euhydration; HY, hypohydration. * $P < 0.05$ from control conditions.

the muscle pieces were weighed and hydrolyzed in 0.5 ml of 2 N HCl. After neutralization with 1.5 ml of 1 N NaOH, glucose concentration was determined by a standard enzymatic fluorometric technique (23). Blood samples were collected in syringes containing lithium-heparin. The blood samples were immediately analyzed in triplicate for hemoglobin concentration (Coulter hemoglobinometer), plasma protein concentration (American Optical refractometer), and osmolality (Advanced Instruments microosmometer).

Statistical analyses. Statistical comparisons were made by a repeated measures analysis of variance. If significant main effects were indicated, Tukey's critical difference was calculated to locate significant differences at $P < 0.05$. All data are presented as means ± SE.

RESULTS

Table 1 presents the body weight (baseline) and blood constituent data obtained during the morning after 15 h of rest (T2) during EU and HY trials as well as in the control condition. Despite drinking the required fluid volumes, the subjects had reduced body weights (1.3%; $P < 0.05$) and increased hemoglobin ($P < 0.05$) from control values. These findings were not viewed as a clear indication of a reduced body water content, because plasma protein content and plasma osmolality were not different between EU trial and control conditions. In the HY trial, the subjects maintained a 4.8% reduction ($P < 0.05$) in body weight (below baseline) during the 15-h overnight rest. Values for hemoglobin, plasma protein content, and plasma osmolality were significantly greater in the HY trial than in EU trial and control conditions.

Table 2 presents the water content of the muscle samples obtained immediately after 2 h of intermittent cycle ergometer exercise and on the following morning 15 h after the carbohydrate meal. Muscle water content was 8% lower ($P < 0.05$) at T1 in the HY trial than at T1 in the EU trial; muscle water content was 8% further reduced ($P < 0.05$) at T2 compared with T1 in the HY trial.

TABLE 2. *Muscle water content after 2 h of intermittent cycle exercise and after 15 h of overnight rest*

	T1	T2
EU	313±8	301±4
HY	288±9*	265±5*†

Values are means ± SE in ml/100 g dry wt; n = 8 men. T1, after 2 h of intermittent cycle exercise; T2, after 15 h of overnight rest. * $P < 0.05$ from Eu conditions. † $P < 0.05$ from corresponding value at T1.

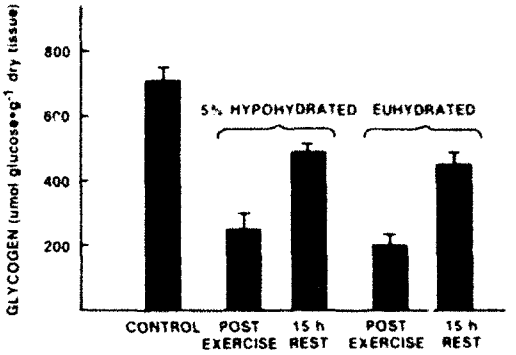

FIG. 2. Muscle glycogen concentration from samples obtained after 2 h of intermittent cycle exercise and the following morning 15 h after carbohydrate meal was ingested under both euhydrated and hypohydrated conditions. Normal glycogen concentration from samples obtained on a separate day is also given.

Muscle water content was ~13% lower ($P < 0.05$) at T2 during the HY trial than at T2 during the EU trial. Although muscle water content tended to be lower at T2 than at T1 in the EU trial, the difference was not significant.

Figure 2 presents the muscle glycogen concentrations obtained during both experimental conditions. For both trials, muscle glycogen concentration was reduced ($P < 0.01$) at T1 (postexercise) compared with control values, and no difference was found between EU and HY trials. Muscle glycogen concentration increased ($P < 0.05$) between T1 and T2 during both trials, and the amount of muscle glycogen resynthesis was similar during EU and HY trials (251 ± 32 and 239 ± 30 μmol glucose/g dry tissue, respectively).

DISCUSSION

Exercise in the heat resulted in a substantial (~3.5-liter) body water loss that corresponded to ~5% of body weight. During the EU trial, the subjects were ~1% below their baseline body weight, but the difference was not significant and falls within the definition of euhydration (9). The slightly reduced body weight was probably the result of reduced food intake during the previous 36 h and not indicative of reduced body water content. During the EU trial, plasma protein content as well as osmolality values were not different from the control values, again suggesting that the subjects were euhydrated. Exercise- and heat-induced hypohydration should result in a hyperosmotic hypovolemia (27, 28). The increased plasma osmolality and elevated hemoglobin and plasma protein content values indicated a hyperosmotic hypovolemia similar in magnitude to that normally expected with a 5% reduction in body weight or an 8% reduction in total body water (27).

The present study investigated whether muscle glycogen resynthesis during a 15-h recovery period after exercise is impaired in individuals who have incurred a body water deficit. This question is particularly relevant when it is considered that, in humans, rehydration by voluntary fluid intake is a fairly slow process and may take several days in the absence of a meal. Moreover, fluid replacement may be further compromised under certain industrial or military conditions in which water availabil-

ity is limited. Muscle glycogen resynthesis during the 15-h recovery period was about the same in both experiments, averaging 16.3 μmol glucose · g dry tissue^{-1} · h^{-1} (~4.1 μmol glucose · g wet wt^{-1} · h^{-1}). Recently, Blom et al. (2) have reported that ingestion of 0.35 g glucose/kg body wt immediately after exercise and again 2 and 4 h later resulted in a glycogen resynthesis rate of 2.1 μmol glucose · g wet wt^{-1} · h^{-1}. Increasing the glucose intake at each time period to 0.70 g/kg body wt resulted in a 5.8-μmol glucose · g wet wt^{-1} · h^{-1} synthesis rate, with no further increase observed when greater amounts of glucose were consumed. Thus, in their subjects, postexercise muscle glycogen resynthesis was maximized with the ingestion of ~156 g carbohydrate over a 6-h period. In the present study, a single 400-g carbohydrate meal was given to each subject. Based on body weight, this was equivalent to a carbohydrate intake of 5.5 g/kg body wt with a range of 4.2 to 6.5 g/kg body wt. Therefore it is likely that carbohydrate intake did not limit glycogen resynthesis rate, at least during the initial 4–6 h of recovery.

In this study, ingestion of the carbohydrate meal was delayed by ~7 h after strenuous exercise because of the hydration requirements of the experiments. This raises the possibility that some resynthesis may have occurred during this time period. Several reports suggest that a small but significant amount of synthesis occurs during the first 2 h of recovery from exercise under fasting conditions (12, 13, 18, 24). However, after 2–4 h of recovery, glycogen concentration in the previously active muscle stabilizes as long as fasting continues (13, 18). Maehlum and Hermansen (18) extended this observation through 12 h of fasted recovery. Peters Futre et al. (24) have also demonstrated that resynthesis during the initial 90 min of recovery is not impeded by light-intensity exercise. Subjects in the present study, on completion of the 2-h cycling bout, rested for ~2.5 h and then completed several hours of passive heat exposure with occasional 5- to 10-min bouts of low-intensity upper body exercise in the heat. Thus our concern was for further glycogen loss, and, as such, we instructed the subjects to minimize activity of the previously active quadriceps muscle groups. Although changes in muscle glycogen content during the ~7 h before the carbohydrate meal cannot be discounted, synthesis likely did not occur to any significant extent.

Estimated muscle water content values during the EU trial (313 and 301 ml/100 g dry wt) are similar to values reported by others (30, 31). Sjogaard and Saltin (30) found that total muscle water content was similar in different muscles (soleus, vastus lateralis, gastrocnemius, and triceps brachii), averaging 320 ml/100 g dry wt. Young et al. (31) reported that vastus lateralis water content ranged from ~330 ml/100 g dry wt in resting individuals to ~350 ml/100 g dry wt immediately after 30 min of cycle exercise. It is interesting to note that, during the HY but not the EU trial, muscle water content decreased significantly from T1 to T2 (Table 2). This suggests that water loss from previously active muscle may continue during recovery periods, particularly when the individual is hypohydrated. This observation is in agreement with observations of Nose et al. (21), who demonstrated that thermal dehydration primarily pulled fluids from skeletal muscle to help defend plasma volume. During hypohydration, reductions in muscle water are also probably accompanied by altered electrolyte concentrations (29); the combined effects could disrupt the membrane electrochemical potential and thereby contribute to muscle fatigue with subsequent exercise (28).

In summary, the results of this study demonstrate that muscle glycogen resynthesis during the recovery from exercise is not limited by a moderate reduction in body and muscle water content. The water stored with glycogen probably does not have a regulatory function per se. Rather, increases in muscle water content are likely a consequence of an osmotic drive induced by increased glycogen granule content in the sarcoplasm of the muscle cells. Thus increases in glycogen concentration and tissue water content parallel one another under euhydrated conditions. With hypohydration, however, muscle water content remains reduced despite continued muscle glycogen resynthesis, suggesting that intracellular osmolality is insufficient to overcome the hypohydration-mediated osmotic drive to defend plasma volume. Although the possibility exists that a more severe reduction in muscle water content might influence glycogen resynthesis, the findings of this investigation appear to indicate that water content is not a critical factor for the restoration of muscle glycogen after heavy exercise.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision unless so designated by other official documentation. This study has been approved for public release; distribution is unlimited.

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